Definition
Albeit it is somewhat controversial as to whether subclinical hypothyroidism exists, technically subclinical thyroid disease (SCTD) is defined as serum free T(4) and free T(3) levels within their respective reference ranges in the presence of abnormal serum TSH levels. Most often, patients present with vague, nonspecific symptoms that are suggestive of hypothyroidism, but on many occasion, attempts to identify patients clinically, via laboratory values, have not been successful – conventionally, at least.¹

Epidemiology
Depending on the source, the prevalence ranges from 4% to 15%.² From 1988 to 1994 (I could not locate more current data), in the US National Health and Examination Survey (NHANES III), excluding known thyroid disease, 4.3% of 16,533 people had subclinical hypothyroidism. As we age, prevalence increases. It is present more often in females than males, and lower in blacks than in whites.³

Etiology⁴
The causes of subclinical hypothyroidism are the same as those of overt hypothyroidism.

Most patients have Hashimoto’s thyroiditis with elevations of antithyroid peroxidase antibodies (anti-TPO). Other major causes include prior ablative or antithyroid drug therapy for Graves’ disease; prior partial thyroidectomy; radiation therapy with Hodgkin lymphoma, leukemia, or brain tumors; inadequate T4 replacement therapy for overt hypothyroidism; and drugs impairing thyroid function.⁵

Diagnosis
Diagnosis is based on blood tests. It may occur with the presence or absence of mild symptoms of hypothyroidism.

In my opinion and experience, to increase the precision of the diagnosis, serum TSH, FT3, and FT4 should be tested. However, in circumstances where there is a strong indication for T4 therapy, such as pregnancy or infertility, T4 and/or T3 replacement should be initiated if TSH is elevated and/or the individual is symptomatic.

Consequences of Subclinical Hypothyroidism
A substantial proportion of patients will eventually develop overt hypothyroidism. Studies have shown in 10 to 20 years of follow-up, the cumulative incidence of overt hypothyroidism ranges from 33% to 55%.⁶⁻⁷

Subclinical hypothyroidism has been associated with an increase in cardiovascular risk factors, markers of inflammation, vascular reactivity, endothelial function, and carotid intima media thickness.⁸⁻¹⁰ Some subjects have been observed to have diastolic dysfunction, along with increased peripheral vascular resistance.¹¹

Other comorbidities may also exist. For example, in a cross-sectional study, nonalcoholic fatty liver disease (NAFLD) was correlated with serum TSH levels. Thirty percent of individuals had ultrasonographic findings of NAFLD (versus 20% of controls), while 20% had abnormal liver enzymes.¹²

Management
Virtually all experts recommend treatment with serum thyrotropin (TSH) concentrations >10 mU/L. The routine treatment of asymptomatic patients with TSH values between 4.5 and 10 mU/L remains controversial.¹³

Some groups suggest treatment in patients with subclinical hypothyroidism and TSH levels greater than 10 mU/L, given the data linking atherosclerosis and myocardial infarction, along with increased risk of progression to overt hypothyroidism.

There are few reported data showing benefit or harm of thyroxine (T4) treatment in patients with TSH values between 4.5 and 10 mU/L. A clinical consensus group (comprising representatives from the Endocrine Society, American Thyroid Association (ATA), and the American Association of Clinical Endocrinologists) did not recommend routine treatment for such patients, but recommended monitoring TSH levels every 6 to 12 months.¹⁴

Treatment Goals¹⁵
The goal of therapy is to reduce the patient’s serum TSH concentration into the normal reference range, as well as improve symptoms. 1.4 mU/L is the mean serum TSH for the general US population, with 90% having serum TSH levels <3.0 mU/L. Many experts recommend a therapeutic TSH target of 0.5 to 2.5 mU/L in young and middle-aged patients, while a TSH target of 3 to 5 mU/L may be appropriate in patients over age 70 years.

Integrative and Holistic Approach
Throughout my training as a naturopathic doctor, I was indoctrinated with “don’t treat the numbers, treat the patient.” Typically in our view, subclinical hypothyroidism is a mild elevation in TSH (this value varies amongst various CAM providers, but typically ≥2.5 µU/L/
ml, but less than 10 µU/ml), but may also be based more on clinical symptoms.15 These patients don’t meet the criteria for hypothyroidism via standard hormone tests per se (i.e., free or total T3 and free T4), but yet present clinically as hypothyroid.16

The question then arises, to treat or not to treat with thyroid hormone? I believe this needs to be taken on a case-by-case basis, but studies do show that patients generally have an improved sense of well-being, and measurable lipid and cardiac abnormalities tend to improve.17,18 For those with thyroid autoantibodies, it may also prevent progression of the autoimmune process with thyroid replacement.19

In my opinion, investigation of other organic etiologies that overlap hypothyroid symptomatology should be excluded first, before initiating thyroid hormone replacement. Iron deficiency anemia, hypercortisolism, and adrenal hypofunction are just a few examples.

DHEA-S looks at adrenal function, and the stress hormone cortisol (secreted from the adrenal cortex) inhibits T4 to T3 conversion.20 T4 to T3 also need cofactors of iron (Fe), zinc (Zn), methylcobalamin (B12), and selenium (Se) to convert.21 If FT3 is low or low normal, while FT4 is normal, you might consider a cofactor conversion issue. In order for thyroid hormone to be functionally produced, tyrosine and iodine also need to be present.22

Once I have ruled out iron deficiency anemia, metabolic syndrome, diabetes and frank hypothyroidism, and the diagnosis “subclinical hypothyroidism” is determined, I institute the following treatments before using thyroid hormones. I have seen improvements in TSH, FT4, and FT3 values in over 100 patients, and more importantly, improvement in most if not all of the patient’s health concerns.

- proper sleep hygiene
- stress management
- exercise
- contrast hydrotherapy (water therapy) to regions over the thyroid and suprarenal glands
  - Consists of 3 min hot, 30 seconds cold, in sets of three, three times daily. Always ends on cold.
  - The theory is that this acts as a pumping mechanism and stimulates the glands.
- high-potency multivitamin/mineral combination, including RDA of iodine (varies from 90 mcg to 290 mcg depending on age, pregnancy, and lactation)23
  - Provides cofactors for T4/T3 conversion.
- adaptogenic botanical medicines (beyond the scope of this discussion)
  - These have traditional use, as well as evidence regarding their efficacy to assist the body’s ability to “adapt” to stress, improve stamina, energy, and mood.24–26
- DHEA supplementation if DHEA-S is low or normal for age and gender27

Conclusion

Subclinical hypothyroidism is becoming increasingly prevalent in the US, especially when one considers the ubiquity of endocrine disruptors in our environment, role of chronic stress, and poor dietary choices.28 Many patients seek out integrative/CAM providers because they don’t feel listened to and/or their symptoms may be brushed off and ignored.

This article has (hopefully) opened the door to the view that this may be an overlooked etiology for a patient’s health concerns, and appropriate treatment may improve not only laboratory values but quality of life.

Notes

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When he’s not working, Dr. Born enjoys spending time with his wife and son, being in the great outdoors, reading, writing, traveling, and playing with his three rescued Persian cats.